Amendments to the Claims:

Claim 1. (Currently Amended) A method for identifying a candidate modulatory compound for ameliorating or delaying an impaired glucose tolerance condition~~, atherosclerosis, or obesity~~, said method comprising:

(a) providing a *C. elegans* or isolated *C. elegans* cell expressing a gene that encodes a polypeptide having at least 85% homology to SEQ ID NO:54 and that functions in insulin signaling; and

(b) contacting said *C. elegans* or isolated *C. elegans* cell with a candidate compound, wherein a decrease in expression or activity of said gene following contact of said *C. elegans*, or said isolated *C. elegans* cell with said candidate compound identifies a candidate modulatory compound for ameliorating or delaying an impaired glucose tolerance condition~~, atherosclerosis, or obesity~~.

Claims 2 and 3 (Canceled).

Claim 4. (Previously Presented) The method of claim 1, wherein said gene is a nematode *daf-16* gene.

Claims 5-11 (Canceled).

Claim 12. (Currently Amended) The method of claim 1, wherein said ~~gene~~ polypeptide has at least 90% amino acid sequence identity to SEQ ID NO:54.

Claim 13. (Previously Presented) The method of claim 1, wherein said gene is a human gene.

Claims 14 and 15 (Canceled).

Claim 16. (Currently Amended) The method of claim 1, wherein said ~~gene~~ polypeptide has at least 95%-amino acid sequence identity to SEQ ID NO:54.

Claim 17. (Currently Amended) A method for identifying a candidate modulatory compound for ameliorating or delaying an impaired glucose tolerance condition~~, atherosclerosis, or obesity~~, said method comprising:

(a) providing a *C. elegans* or isolated *C. elegans* cell expressing a gene that hybridizes under highly stringent conditions to the complement of a nucleic acid sequence encoding the sequence of SEQ ID NO:54 and that functions in insulin signaling;

(b) contacting said *C. elegans* or isolated *C. elegans* cell with a candidate compound, wherein a decrease in expression or activity of said gene following contact of said *C. elegans* or said isolated *C. elegans* cell with said candidate compound identifies a

candidate modulatory compound for ameliorating or delaying an impaired glucose tolerance condition~~, atherosclerosis, or obesity~~.

Claim 18 (Canceled).

Claim 19. (Currently Amended) The method of ~~claim 18~~ claim 17, wherein said ~~human~~ gene is AFX

Claim 20. (Currently Amended) The method of ~~claim 18~~ claim 17, wherein said ~~human~~ gene is FKHR.

Claim 21. (New) A method for identifying a candidate modulatory compound for ameliorating or delaying an impaired glucose tolerance condition, said method comprising:

(a) providing a *C. elegans* or isolated *C. elegans* cell expressing a human FKHR gene; and

(b) contacting said *C. elegans* or isolated *C. elegans* cell with a candidate compound, wherein a decrease in expression or activity of said FKHR gene following contact of said *C. elegans* or isolated *C. elegans* cell with said candidate compound identifies a candidate modulatory compound for ameliorating or delaying an impaired glucose tolerance condition.

Claim 22. (New) A method for identifying a candidate modulatory compound for ameliorating or delaying an impaired glucose tolerance condition, said method comprising:

(a) providing a *C. elegans* or isolated *C. elegans* cell expressing a human AFX gene; and

(b) contacting said *C. elegans* or isolated *C. elegans* cell with a candidate compound, wherein a decrease in expression or activity of said AFX gene following contact of said *C. elegans* or isolated *C. elegans* cell with said candidate compound identifies a candidate modulatory compound for ameliorating or delaying an impaired glucose tolerance condition.

Claim 23. (New) The method of any one of claims 1, 17, 21, and 22, wherein said glucose tolerance condition is atherosclerosis.

Claim 24. (New) The method of any one of claims 1, 17, 21, and 22, wherein said glucose tolerance condition is obesity.